

Editorial

Haemodynamic effects of eating

... Those, who are afflicted with it, are seized, while they are walking, and more particularly when they walk soon after eating, with a painful and disagreeable sensation in the breast, which seems as if it would take their life away, if it were to increase or to continue: the moment they stand still all this uneasiness vanishes.

WILLIAM HEBERDEN (21 July 1768)¹

Cardiologists not infrequently find themselves lecturing at evening meetings on the subject of angina pectoris. Many would welcome the opportunity to round off their presentation, before dinner, with one or two well chosen observations on the haemodynamic stresses of eating. Compared with the effects of exercise, mental stress, and cold (on which the lecturer will have talked with authority), eating presents difficulties because published reports are sparse and inconsistent. The cardiologist's experience may be limited to vague recollections of childhood warnings not to go swimming within an hour of eating a meal for fear of potentially fatal cramps in the exercising muscles robbed of their blood supply by the bloated gut (or was it that redirection of blood to exercising muscles would cause violent indigestion?). Further information about these effects comes from three recent studies, two of which appear on pages 22 and 26 of the current issue.²⁻⁴

The studies

It is reassuring that two articles that rely on widely different methods come to broadly similar conclusions. In a paper published in the *British Heart Journal* of June 1989 Kelbaek *et al* used first pass radionuclide imaging to study left ventricular dimensions before and after a standard Danish meal was eaten.² They showed considerable increases in both the end systolic and end diastolic volumes of the left ventricle in healthy volunteers studied in the supine position. The ejection fraction did not increase; but heart rate rose by 17%, stroke volume by 41%, and cardiac output by 62%. The blood pressure did not change; so eating must have caused at least a 50% drop in peripheral vascular resistance, presumably due to vasodilatation in the mesenteric bed.

The group from Nottingham used respiratory gas exchange to estimate cardiac output and, more ambitiously, extended their observations to include exercise tests before and after a meal with subsequent measurements of peripheral blood flow in the calf and forearm (relevant to the swimming dilemma).³ Again healthy volunteers were studied, but this time they were sitting or standing. The Nottingham meal was marginally more frugal than the Danish one. (It would be interesting and feasible to study the effects of differing meal sizes and compositions—is a snack as bad as a banquet? If not, at what stage does the dose response curve flatten out?) The results showed an increase in cardiac output of 30% after a meal and this differential persisted on exercise. There was a 16% increase in heart rate and no change in blood pressure. There were significant increases both before and after

exercise in measures of work done and metabolic activity after eating. Total peripheral vascular resistance fell. Limb blood flow, before and after exercise, however, was not reduced by eating; and since it seems unlikely that other vascular beds became vasoconstricted after food Yi *et al* concluded that eating was haemodynamically stressful. They expressed surprise that more patients with angina and heart failure do not describe a deterioration of their symptoms after a meal. Some clinicians (and that includes me) might agree with William Heberden¹ that angina on exercise after eating is, in fact, quite common. As for deterioration in heart failure, perhaps some parallels can be drawn here between the substantial food-induced drop in peripheral vascular resistance shown in all three studies and the action of the currently fashionable drugs used for the treatment of this condition. Indeed, just such an action is highlighted by Herrlin *et al* whose study of patients with heart failure appears in this issue.⁴ The qualitative effects of eating and angiotensin converting enzyme inhibition on cardiac output, systemic vascular resistance, and pulmonary capillary wedge pressure are broadly similar.

Questions raised

Like all good studies, these three reports raise many questions. It would be helpful to sort out the mechanisms of these circulatory adjustments. Although plasma catecholamines did not vary before and after food in the Danish study,² reference is made to the effects of the autonomic nervous system. Fasting suppresses catecholamine production, presumably to conserve calories, whereas feeding activates the sympathetic system.⁵ In an earlier study, similar to their current one, Kelbaek *et al* showed that the stroke volume and heart rate increased after a meal both at rest and on upright exercise. The increase in stroke volume, but not heart rate, was inhibited by autonomic blockage with metoprolol and atropine. Plasma concentrations of adrenaline were unaffected by eating, so these workers concluded that parasympathetic withdrawal may be important in the postprandial changes that they observed.⁶ Hormonal factors may also have a role—for example infusion of vasoactive intestinal polypeptide causes a considerable reduction of peripheral vascular resistance, drop in blood pressure, and compensatory tachycardia. Cardiac output rises considerably.

Whatever mechanisms underlie these interesting haemodynamic changes, clinicians will be anxious to see whether they are affected by the altered conditions associated with disease states. Published reports are inconsistent, possibly because of differences in meal composition and the mode of exercise (upright/supine). One study confirmed the increase in heart rate after a meal in subjects with postprandial angina, who also had a larger blood pressure response on exertion.⁷ Angina developed more rapidly when these patients were exercising after a meal. The double product (systolic blood pressure \times heart rate) at the onset of pain was the same before and after the meal. This strongly suggests that the harmful effects of

eating are more likely to be the result of haemodynamic stress rather than the diversion of blood away from the coronary arteries to other vascular beds.

It would be interesting to compare the haemodynamic effects of eating in angina patients whose symptoms are or are not aggravated by food. How are these effects modified by β blockers, prophylactic nitrates, and different calcium antagonists? Theoretically β blockers should be helpful whereas food might attenuate the value of nitrates.

Some data have been published on the haemodynamic effects of eating in patients with left ventricular failure. In one such study, heart rate and cardiac output increased after eating, but mean blood pressure fell.⁸ In another study the ejection fraction in patients with moderate ventricular dysfunction increased after a meal.⁹ The observed effects did not seem to be particularly detrimental and were similar to those reported in the current study by Herrlin *et al.* These changes were accentuated by angiotensin converting enzyme inhibition but were also quite considerable in a group treated with placebo. Any study of vasodilator treatment that does not take account of these basal fluctuations is likely to be seriously flawed.

Advice

All patients with angina should be advised to avoid exertion after eating. Perhaps an hour is sufficient (the current observations were made at 30 minutes) though haemodynamic changes have been seen as long as six hours after a meal. Significant changes were still present 2–4 hours after eating in heart failure patients treated with placebo. Further information is needed before drug treatment can

be tailored to a specific requirement and appropriate advice given on the size and content of the meal least likely to provoke angina. Meanwhile, the well informed lecturer might conclude by inviting the audience to reflect on the negative inotropic effect that healthy people show after drinking alcohol.¹⁰

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